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Modern Concepts of Cardiovascular Disease

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LOCALIZATION OF CARDIAC INFARCTS

Without doubt cardiac infarction has been a major cause of death for centuries although it was first accurately described by Herrick only twenty-seven years ago. Since then progress in the localization of infarcts has been rapid. Parkinson and Bedford, Wilson, Wood and Wolferth, and others were able to differentiate in many cases between anterior (Q_3T_1) and posterior (Q_1T_3) types of lesions. Further refinements of diagnosis and of prognosis have been made possible by observing the characteristic distribution of the coronary arteries to the individual muscle bundles of the heart.

Five centuries of anatomists, since Vesalius, have noted the complex structure of the ventricular walls. MacCallum and Mall gave names to the several muscle bands. Their observations were extended by Robb and Robb to include the vascular and Purkinje supply to, and the functioning of, those muscles. Seventy-seven clinical examples (complete with history, E.C.G. and autopsy) elucidate the disease syndromes peculiar to each.

First let us note that the ventricular walls are composed of circular bands (the deep sinospiral and bulbospiral muscles). These are thinly covered by diagonal fibres (the superficial sinospiral and bulbospiral muscles) which converge at the apex, turn in, and pass back as the longitudinal (papillary and pectinate) fibers to line the interior of the ventricles and to insert on the auriculoventricular valves. The two superficial muscles are innervated from their papillary ends by the anterior and posterior divisions of the left branch of the Bundle of His. The circular muscles are innervated by penetrating branches that emerge "radially" from the Purkinje system to the proper level and there turn to run parallel with the circular fibers. The Purkinje tissue is not merely a subendocardial layer, but is a profuse myocardial network ramifying in many planes and extending *nearly* to the epicardium. There is free communication between the peripheral extensions of the right and left bundle branches both in the outer walls of the heart and in the septum. If the action current is conducted along the system, then presumably it is conducted in all of the system and not merely in those fibers which penetrate radially from endocardium to epicardium. It is apparent that a radial penetration theory would be primarily applicable to the innervation of the circular muscles and would be of lesser validity in the description of the spiral muscles.

Let us now consider the four easily recognizable ventricular muscles in detail:

I. The Superficial Sinospiral muscle (S.S.S.) arises broadly from the right base, curves forward around the acute margin of the right ventricle and converges at the apex as the anterior horn, to plunge deeply and form the anterior papillary muscle, inserting chiefly upon the anterior mitral valve of the left ventricle. The right sheet-like portion of this muscle is supplied by superficial twigs from descending branches of the right coronary artery. (This region is rarely infarcted.) The apical part of this muscle is supplied by the more distal side branches of the left anterior descending coronary artery. This muscle is innervated by the anterior division of the left branch of the Bundle of His, which runs directly to the anterior papillary muscle, there forming a rich anastomosis about its fibers, and following those fibers to the surface of the heart. Its function is fixation of the anterior mitral leaflet during systole.

Infarction of this muscle alone would ensue from obstruction in the distal collateral branches of the left anterior descending artery. Clinically we note much pain, a slightly dropping blood pressure for several days, occasionally a friction rub, and an excellent prognosis. In such cases as came to autopsy death was caused by cerebral hemorrhage, nephritis, etc. The electrocardiogram shows a slight R-T elevation in all leads, which places it in the "anterior" category. This is anatomically correct with respect to the larger portion of this muscle.

II. The Superficial Bulbospiral muscle (S.B.S.) arises around the left base, proceeds anteriorly

IMPORTANT ANNOUNCEMENT!

The Fourth Edition of the "Nomenclature and Criteria for Diagnosis of Diseases of the Heart" will be ready September 15. The book has been completely revised and now includes "An Outline for the Pathological Diagnosis of Cardiovascular Diseases and Anomalies." It is profusely illustrated and contains more than twice as many pages as the last edition. The price is $2.00 per copy, postpaid.

All orders, with remittance, should be sent to the American Heart Association, 50 West 50th Street, New York, N. Y.

across the cephalic two-thirds of the left ventricle, covers much of its obtuse lateral border and almost all of the diaphragmatic surface of both ventricles. Spiraling downwards it forms a compact band which contributes to the papillary muscles at the right apex, encircles the left apex, penetrates deeply as the posterior horn of the left apex and constitutes the lower septum and the papillary muscle to the inferior mitral valve. The apical and papillary portions are supplied by collaterals of the left anterior descending artery (proximal to those aforementioned branches entering the anterior horn of the S.S.S.), and by the distal portions of the left obtuse marginal artery, the accessory posterior left ventricular and the posterior descending coronary arteries. The large posterior division of the left branch of the Bundle of His runs directly to the papillary portion of this muscle. Its function is to fix the inferior mitral leaflet during systole. When damaged, the signs are similar to those of S.S.S. infarction except that one less often hears a friction rub. The prognosis in experimental animals is as good as for the S.S.S. muscle. In patients the prognosis may be slightly more serious if the lesion results from a progressive condition which has first occluded the distal portion of the left anterior descending and is progressing to an intermediate portion. Experimental infarction of this muscle causes the S-T in lead I of the electrocardiogram to become depressed and that in lead III to become elevated. If the septal portion is involved Q_3 will be present. This muscle is mainly posterior in its distribution and the E.C.G. change is the "posterior" type. It is possible to have this E.C.G. with the lesion in the anterior band where it encircles the apex. This type of infarction is common, patients usually surviving several attacks. Half of such cases coming to autopsy died of another cause.

III. The Deep Sinospiral muscle (D.S.S.) is a powerful transverse constrictor, deficient at base and apex, encircling both ventricles and forming most of the septum. The right portion is supplied by the descending branches of the right circumflex coronary artery; the septal portion is supplied by the penetrating branches of the anterior and posterior descending arteries and by the superior septal branch of the right coronary artery. The left portion is supplied through the descending branches of the left coronary and especially by the large first lateral collateral of the left anterior descending artery. The muscle has Purkinje supply from both the right and left branches of the Bundle of His, the right being almost exclusively distributed to it. Experimental infarction of either the right or left portion produces a marked fall in blood pressure, numerous premature beats, and considerable shock. The function is to maintain the lesser circulation and also to cause the ejection of blood into the greater circulation early in systole. The RS-T_1 is elevated and RS-T_3 is depressed after injury to this muscle. Half of the autopsied cases showed infarcts in the anterior pathway and would have been diagnosed as "anterior" lesions. The remainder were lateral, posterior, or septal, and would have been difficult to localize by the "T_1" method. These patients had greater fall of blood pressure, more shock, often with atypical or little pain. Generally there are fewer attacks of infarction in this muscle before death occurs. All cases coming to autopsy died because of the infarction, after periods ranging up to about two weeks.

IV. The Deep Bulbospiral muscle (D.B.S.) is a heavy sphincter extending around the upper third of the left ventricle, surrounding the mitral and aortic orifices. It is supplied by penetrating branches of the left circumflex artery. The Purkinje supply is entirely from the left branch of the Bundle of His. The function of this muscle is to finish the emptying of the left ventricle and to maintain pressure at the end of systole. When it relaxes, the aortic valves close. Infarction causes the blood pressure to fall abruptly to very low levels. Ventricular tachycardia is common. Immediate mortality in experimental animals is about 60%. Among 1000 electrocardiograms of autopsied cases only three of this type, with marked elevation of R-T in all leads, were found. One of these patients had an attack of pain at 8 a.m., the systolic blood pressure fell from 190 to 100 at once and continued to fall, the pulse was totally irregular, death occurring in five hours. Another patient was admitted to the hospital for heat prostration. Two days before death there was "substernal pressure." At that time the E.C.G. showed myocardial damage but not infarction. On the day of death, even as an E.C.G. was being taken the end came. The record showed a somewhat lowered voltage in all three leads with a high RS-T take-off in all. The third patient had chronic rheumatic endocarditis with decompensation for which she had been hospitalized several weeks. She suddenly complained of "pain between the shoulders." The heart rate dropped to 27 but presently quickened, the blood pressure fell abruptly, pulmonary edema and prompt death ensued. The infrequency of this type of E.C.G. together with the experimental data, and the rapidity of death recorded in these instances lead to the conclusion that this is the most severe type of lesion and that it is rarely observed because individuals die before E.C.G.'s can be obtained.

Anatomically complex lesions are not uncommon. Progressive coronary sclerosis may involve one muscle after another, and the resultant electrocardiograms represent an algebraic summation of the upward and downward displacements. Thus extensive lesions may balance out to produce low voltage.

The "left lateral ventricular infarcts" of Wood and Wolferth are of the complex type, because for example occlusion of the left obtuse marginal artery must affect three muscles. Conduction disturbances will ensue if occlusion is proximal to the septal branch of the left anterior descending artery, because it (together with the posterior branch of the right coronary artery) supplies the Bundle of His. Injury to the S.A. node with auricular fibrillation is often due to interference with the left circumflex artery although its main supply is from the right coronary.

Summary

There is no antagonism between "anterior or posterior" and "muscle bundle" localization. The former is valuable when infarcts lie in the more common localities; the latter extends analysis to more unusual localities and rests on an anatomical and physiological basis. It has been applied to 77 cases proven at autopsy.

As a "thumb-nail" diagnostic method, one may ignore Lead II and note that R-T_1 depression indicates involvement of the S.B.S. muscle; if R-T_3 is depressed the D.S.S. is implicated and the prognosis is guarded. An S.S.S. lesion raises both R-T_1 and R-T_3 above the iso-electric level. R-T take-off from or near the peak of R in all leads, especially in the presence of low voltage, indicates a D.B.S. lesion, and here the prognosis is most serious. Treatment of D.S.S. and D.B.S. lesions should be much more prolonged and effort more curtailed than for lesions of the superficial muscles.

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